Cerebrovascular Occlusive Disease

Experience with Panarteriography in 300 Consecutive Cases

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■ *Three hundred patients with cerebrovascular occlusive disease have had cerebral angiographic examination at the Veterans Administration Hospital, San Francisco, in the last five years. The present technique consists of preliminary visualization of the aortic arch and the major extracranial branches, followed by selective study of the subclavian and carotid arteries as necessary for evaluation of the intracranial circulation.*

Nine major complications occurred (an over-all incidence of 3 per cent). Two patients died after angiography and seven had major neurologic deficits persisting for more than 24 hours. Three of these patients had permanent damage, but four recovered completely.

One-third of the patients had extracranial disease and one-third had intracranial disease. No significant lesion was found in the remainder. In the 212 patients with lesions, multiple lesions were common, the average number being three. Six patients had brain tumors and five had aneurysms.

The mechanism of the stroke could be ascertained readily in most of the patients, but the extent of the disease and the resulting symptoms varied considerably. Several patients with occlusion of most of the cerebral vessels had minimal symptoms, while others had catastrophic symptoms but only minimal findings at arteriography.

CEREBROVASCULAR OCCLUSIVE DISEASE is the third leading cause of death, yet our understanding of many of the fundamental aspects of this condition, etiologic and physiologic, still is incomplete. Although at least one-third to one-half of the lesions that cause stroke are located in the neck,[5] the significant etiologic role of the extracranial arteries in stroke has been recognized only since the introduction of arteriography, which permits visualization of the entire cerebrovascular tree.[1,2,8]

Since July 1960, we have been using cerebral arteriography frequently in patients thought to have cerebrovascular disease. Our experience with the first 100 patients in whom cerebral angiography was used for diagnosis of stroke has been reported.[1] Our present series is made up of 300

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consecutive cases, including those in the first report. In all of these patients several vessels were examined, since study of individual vessels is usually inadequate in the evaluation of patients with stroke. In this study we measured the risks involved in panarteriography against the benefits, to assess the value of the procedure.

Technique

The details of the technique of examination of individual cerebral vessels have been reported.[1] To obtain maximum information about the cerebrovascular tree while avoiding excessive discomfort to the patient and minimizing the risks involved in injection, the vessels should be studied in an orderly sequence.

The usual method of examination at present consists of retrograde percutaneous catheterization of the brachial artery. Catheters are passed directly into the ascending aorta. Aortic injection gives a good preliminary view of the entire extracranial cerebrovascular system and permits localization of the disease in the extracranial vessels. Needles then can be introduced in the individual arteries as necessary, thus avoiding sites of maximum arteriosclerotic involvement.

An injection of 50 ml of an 80 per cent solution of sodium iothalamate* is made into the ascending aorta under 200 to 300 pounds pressure through a catheter with an internal diameter of 0.046 to 0.063 inches. Multiple exposures are taken at one-second intervals with the patient in the right posterior oblique position. A film measuring 11 x 14 cm is positioned so the lower margin is at the sternal angle. A second injection is made in the anterior-posterior projection. The film extends from the level of the take-off of the vertebral artery to the top of the cranium. If these films are satisfactory, the catheter is withdrawn into the right subclavian artery and 30 ml of a 60 per cent solution of methyl golucamine diatrizoate† is injected into the subclavian artery two to three cm distal to the origin of the vertebral artery. Multiple films are taken in the Towne position for intracranial visualization of the vertebral-basilar system. If contrast is adequate in the intracranial portion of the right carotid system by retrograde filling of the innominate artery, the injection is repeated with the film exposed in the lateral position, which completes the examination of the right carotid artery.

*Angio-Conray, a product of Mallinckrodt Chemical Works, St. Louis.

†Renografin-60, a product of E. R. Squibb & Sons, New York.

Percutaneous puncture of the left common carotid artery is then performed and anterior-posterior and lateral carotid arteriograms are obtained. If visualization of the right carotid system is inadequate, direct injection of the right carotid artery is necessary. Similarly, inadequate visualization of the basilar system from the right vertebral injection requires left vertebral arteriograms obtained by percutaneous catheterization of the left brachial artery.

If this method is not feasible because of the nature of the disease, alternative methods of initial visualization of the aortic arch can be used. If disease in vessels of the upper extremity makes catheterization difficult, the catheter can be passed retrograde from the femoral arteries and the tip can be positioned in the ascending aorta. In this case, both carotid arteries must be punctured percutaneously and at least one study of the subclavian artery should be done to visualize the basilar artery.

Occasionally the patient with extensive cerebrovascular disease has associated arteriosclerosis of the lower extremities. In this instance, an adequate screening examination can usually be accomplished by injection of a contrast agent into one of the great veins in the chest. The best technique for visualization of the aortic arch via the venous route is percutaneous or direct catheterization of the exposed basilic vein in the antecubital fossa or the external jugular vein in the neck. A catheter with an internal diameter of 0.063 inch is passed downward into the superior vena cava near the right atrium. A solution of 80 per cent sodium iothalamate, 1 ml per kg of body weight, is injected under pressure. The patient is told to continue breathing during and immediately after the injection in order to prevent the Valsalva maneuver and obstruction of the flow of dye through the lungs.

Usually, multiple films are made, beginning ten seconds after injection, and six to eight films are taken in the right posterior oblique position two seconds apart. If the degree of contrast in the cerebral vessels is adequate, a similar picture is taken in the anterior-posterior projection as described for direct visualization of the arch.

Complications

Deaths and major complications (Table 1). In the present series, two deaths were directly related to arteriography. The first, in a 70-year-old pa-

TABLE 1.—*Deaths and Major Complications of Panarteriography*

Complication	*Duration and Residual Effects*
Convulsions and cardiac arrest	Resuscitation unsuccessful; patient died 30 minutes after injection
Left hemiparesis and left homonymous field defect	Patient died 3 days after angiography
Hemianopsia and mental deterioration	Permanent mild visual defect and mental impairment
Severe vertigo	Complete recovery in 10 days
Dysarthria, vertigo and lethargy	Complete recovery in 10 days
Dysarthria and mental depression	Recovery, except for slight residual effect, in 10 days
Right hemiparesis	Recovery of leg function after 3 weeks; permanent residual weakness of arm and face
Convulsions and progression of previous left hemiparesis	Permanent increase in spasticity, left side
Convulsions and somnolence	Gradual complete recovery in 3 days

tient, resulted from extravasation of dye after injection of the right subclavian artery near the orifice of the vertebral artery, resulting in temporary closure. The patient had a major stroke and died a week later. At autopsy the right vertebral artery was the only patent extracranial vessel. The second death was in a 76-year-old patient. The only palpable pulse was in the right carotid artery. No vessels were palpable in any of the four extremities. Injection into the right carotid artery was followed by convulsion, temporary anoxia and ventricular fibrillation. Attempts at cardiac resuscitation were unsuccessful. At autopsy all the cerebral vessels were occluded and none of the major coronary arteries were open. Both of these patients had been disabled by their disease. The first had progressive blindness related to retinal ischemia following occlusion of both carotid arteries. The second had progressive loss of vision, episodes of syncope and mental deterioration.

The major complications in this series consisted of cases of major neurologic changes which persisted for more than 24 hours. Vascular occlusions which did not result in neurologic changes were not considered to be major complications since in none of these 300 cases did occlusion of a vessel threaten life or limb. Pneumothorax was not considered a major complication, since it was looked for routinely in the supraclavicular subclavian studies and in all cases of pneumothorax the complication was treated promptly, with negligible patient morbidity. Seven patients had major neurologic complications, but only three of them had a permanent neurologic deficit. The mortality rate in the series was 0.67 per cent (two out of 300) and 2.3 per cent of the patients had major complications (seven of 300), or a total rate of 3 per cent for death and major complication.

Minor complications (Table 2). In 63 cases, intramural or extravascular injections were followed by discomfort to the patient or by difficulty in interpretation of the arteriograms. Significant hematomas occurred after 47 of the individual vessel studies. The minor neurologic complications consisted of 19 instances of convulsion and 18 transient neurologic deficits. All the convulsions occurred immediately following injection and were most common in examinations of the carotid artery. The 18 minor neurologic deficits consisted of neurologic changes which lasted less than 24 hours. These included three cases of transient hemiparesis, two of temporary blindness, three of somnolence, seven of aphasia or dysphasia, and one each of paresthesia, diplopia and vocal cord paralysis.

Pneumothorax occurred in 24 patients, but the only time it posed a serious threat was in a patient with bilateral involvement. Pneumothorax was a frequent complication of supraclavicular subclavian puncture but was relatively infrequent in studies of other arteries. In 14 patients vascular occlusion occurred which lasted more than 48 hours. Twelve of these patients had brachial artery occlusions after retrograde brachial catheterization and two had femoral occlusions after catheterization. In one patient a small fistula of the brachial artery-vein occurred, requiring surgical correction. The 15 miscellaneous complications consisted of nausea, headache, postural hypotension and syn-

TABLE 2.—*Minor Complications of Panarteriography*

Complication	*Number of Patients*
Extravasation	63
Hematoma	47
Minor neurologic deficit	37
Pneumothorax	24
Vascular occlusion*	15
Allergic reaction	8
Miscellaneous†	15

*Includes one traumatic arteriovenous fistula.
†Including nausea, headache, hypotension, syncope.

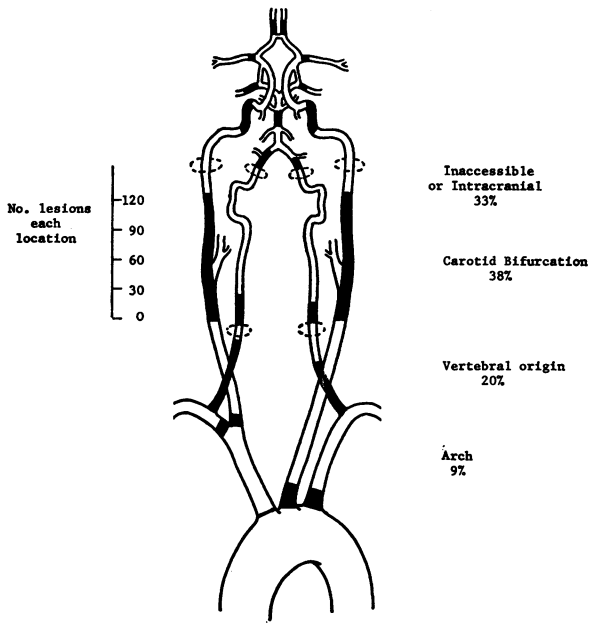

Figure 1.—Location and incidence of significant atherosclerotic lesions. The length of the dark area at each location (measured against the scale at left) corresponds to the number of lesions detected by arteriography in this series.

cope. Only eight patients had minor allergic reactions, consisting mostly of urticaria.

Results

Cerebrovascular occlusive disease was the primary diagnosis before angiography in the 300 patients in this study. The distribution of the 567 lesions considered to be significant is shown (Figure 1). A significant lesion was considered to be one that narrowed the lumen of the vessel by 30 per cent or more. Roughly one-third of the patients had intracranial lesions, one-third had extracranial lesions and in one-third no significant atherosclerotic lesions were visible in the cerebrovascular tree. The 212 patients with definite arteriographic evidence of disease had an average of three lesions each. Eighty-four of the patients with lesions had both intracranial and extracranial disease. Five patients had aneurysms and six had brain tumors.

Visualization of lesions on these arteriograms gave valuable specific information concerning the mechanism of the stroke, including the detection of unsuspected cardiac emboli, emboli from arteriosclerotic plaques or fibrin emboli from proximal ulcerated lesions in the cerebrovascular tree. Also found were atherosclerotic stenosis of vessels, complete atherosclerotic occlusion and hemorrhagic dissection under atherosclerotic plaques with partial or complete occlusion. In many instances the conclusions drawn from these arteriograms were verified at operation or autopsy. These specific findings at arteriography were frequently useful in directing therapy. The specific value of arteriography is demonstrated in the following case reports.

Case Reports

CASE 1. A 54-year-old man had a seven-year history of paralysis of the right side and severe claudication in the legs. He was prematurely senile, had expressive aphasia and his replies were limited to single words. He had right central facial weakness and spastic right hemiparesis which was most severe in the upper extremity. The carotid pulse was weak on the right side and absent on the left. Blood pressure in the right arm was 170/80 mm of mercury and could not be determined in the left arm. Preliminary visualization of the aortic arch by injection of dye into the superior vena cava showed stenosis of the innominate bifurcation, complete occlusion of the right internal carotid artery, complete occlusion of the left common and internal carotid arteries and complete occlusion of the left subclavian artery (Figure 2).

Discussion. The patient's major disability was progressive mental deterioration after a severe cerebrovascular accident seven years previously. If angiography had not been performed it might have been assumed that these changes were solely due to occlusion of the left carotid artery, since no pulse was palpable in this vessel. Most of the patients with generalized deterioration resulting from cerebrovascular disease had simultaneous occlusion of most of the cerebral vessels. In this patient the only patent vessel was the right vertebral artery. In addition, the blood flow in this artery was compromised by stenosis of the innominate artery and blood flow to the brain was further limited since the left vertebral artery provided the principal collateral to the arm. Arteriograms demonstrated that the left subclavian artery filled from injection of the right vertebral artery through retrograde flow in the left vertebral artery; thus the arm was diverting flow from the brain—"subclavian steal."[3,10] After complete angiography revealed the severity of the patient's disease, surgical therapy was directed at improving blood flow in the vertebral arteries. An excellent immediate result was obtained. His aphasia and mental alertness improved noticeably and instead of vegetat-

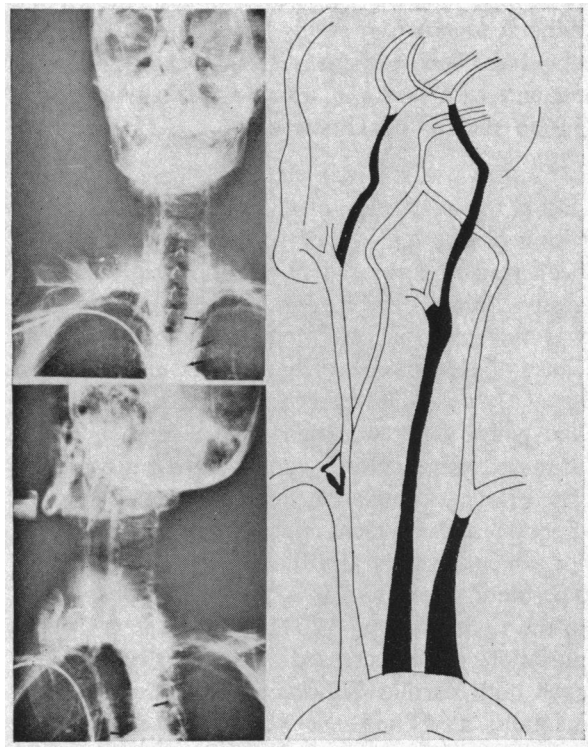

Figure 2 (Case 1).—Venous injection of the aortic arch, showing stenosis of the innominate bifurcation, complete occlusion of the left common and internal carotid arteries and complete occlusion of the left subclavian artery. The left vertebral artery fills retrograde from the right vertebral.

ing in bed he became ambulatory and able to care for himself. Follow-up of one year indicates the initial improvement has been maintained.

Complete cerebral angiography permits the vascular surgeon to select the lesion or lesions most amenable to surgical treatment. We have found the circle of Willis to be hemodynamically competent. Restoration of blood flow in the carotid system will usually relieve arterial insufficiency of the brain stem, and successful surgical treatment of the vertebral artery will improve function in the portion of the brain ordinarily supplied by the carotid arteries.

CASE 2. A 56-year-old man was admitted because of transient hemiplegia, aphasia and sudden onset of vertigo just before admittance. The blood pressure was 170/85 mm of mercury in both arms and there were bilateral carotid bruits. He had right facial weakness and slight weakness of the right arm. Arteriograms showed a plaque at the origin of the left carotid artery and a filling defect in the middle cerebral artery on the same side which resembled an embolic lesion (Figure 3). As a result of these findings long-term anticoagulation therapy was initiated. Follow-up at six months revealed no symptoms or signs of recurrent embolism.

Discussion. Arteriosclerosis is predominantly a disease of large and medium-sized arteries. Most of the catastrophic symptoms occur following sudden occlusion of a previously patent artery. In our experience, many of the occlusions of the middle cerebral arteries found at arteriography or autopsy appear to be of embolic origin and investigation of the neck vessels at operation or autopsy reveal associated ulceration of the corresponding vessel in the neck.[6] These embolic fragments may produce catastrophic symptoms. The associated thrombosis of the vessel occluded by embolus may obscure the nature of the lesion. Other embolic lesions may arise from bits of fibrin which form in irregular areas on the surface of a proximal arteriosclerotic plaque. Subsequent cerebral arteriograms or retinoscopy often reveal that these fibrin emboli have disappeared without a trace.[4,8] This was true in several patients in this series in whom long-term follow-up was possible.

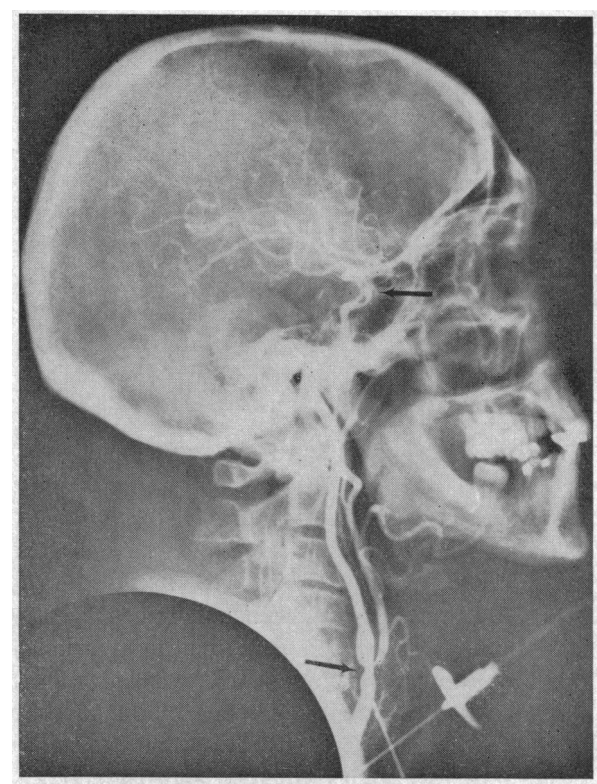

Figure 3 (Case 2).—Proximal atherosclerotic plaque on the origin of the right internal carotid artery with embolization to the distal intracranial portion of the internal carotid artery.

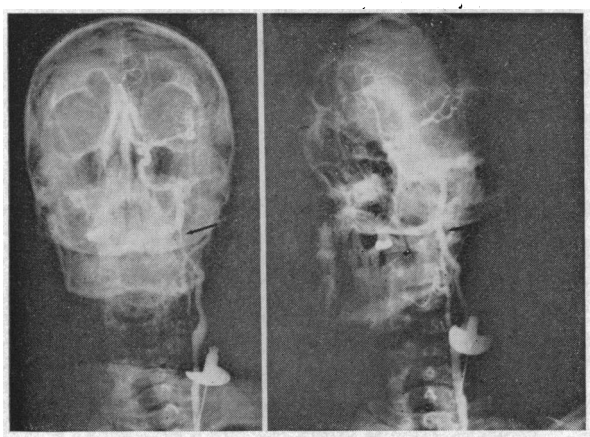

Figure 4 (Case 3).—Left arteriogram taken immediately after onset of right hemiplegia (left frame) shows nearly complete obstruction of the internal carotid artery. The frame at right shows a right arteriogram taken one month later, showing disappearance of the lesion.

CASE 3. A 51-year-old electrician was entirely well until 36 hours before admission, when he had a sudden onset of blindness in the left eye and weakness progressing to complete paralysis of the right arm and leg. The blood pressure was 120/80 mm of mercury in both arms. Pulsations were normal in the neck vessels and he had a bruit over the bifurcation of the left carotid artery. Neurologic examination demonstrated slight residual weakness in the right arm and leg, but significant improvement had occurred since the preadmission examination 24 hours previously, immediately following onset of symptoms. The pressure of the left retinal artery was half that in the right. Arteriograms (Figure 4) showed no abnormality except for nearly complete occlusion of the left internal carotid artery at the base of the skull; follow-up arteriograms a month later showed the artery to have spontaneously returned to normal (Figure 4).

Discussion. The patient recovered almost completely from the stroke although no specific therapy was given. Exploration of similar lesions indicates that acute strokes may be caused by dissection of blood under an atherosclerotic plaque, followed by sudden, complete occlusion of the vessel and distal thrombosis. In this instance the immediate acute obstruction was not complete and permitted some flow of blood past the lesion; the natural fibrinolytic mechanism of the body apparently resulted in reabsorption of the clot and readherence of the plaque to the artery.[9] Follow-up arteriograms a month after the initial episode would have been considered to be "negative." Although it is difficult to prove, it seems likely that such lesions may have been present in a significant number of patients with stroke in whom angiographic examination showed no abnormality.

CASE 4. A 53-year-old truck driver was admitted for evaluation of dizziness and blurring of vision of one and a half years' duration. He had been treated for atrial fibrillation and mild congestive failure since the onset of the symptoms. He was referred for cerebrovascular evaluation because of the absence of blood pressure in the left arm. After detailed questioning he recalled that he had had a slight weakness in the left hand for a year and three episodes of loss of consciousness. The primary complaints were shortness of breath, dyspnea and frequent episodes of ankle edema, for which digitalis and diuretics had been given. The blood pressure was 175/100 mm of mercury in the right arm and 120/100 in the left. Carotid pulsations were decreased and bruits were present over both carotid bifurcations. The patient was mentally alert and the neurologic examination was essentially negative. Four-vessel cerebral arteriography showed complete occlusion of the

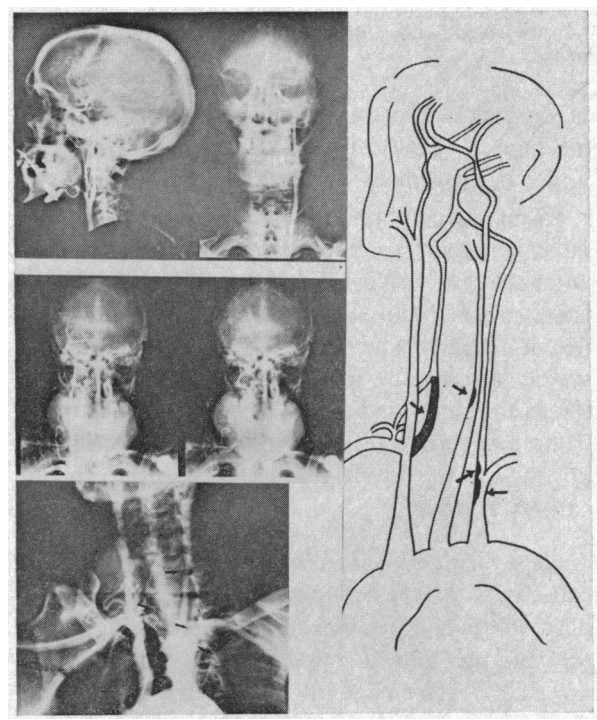

Figure 5 (Case 4).—Standard complete arteriograms. Film at top is arteriogram of left carotid artery; Center film shows right vertebral and carotid arteries and the one at the bottom shows its aortic arch. Together they show complete right vertebral occlusion, a 40 per cent stenosis of the left common carotid artery and pronounced stenosis of the left subclavian and left vertebral arteries.

right vertebral artery, stenosis of the left common carotid artery, almost complete occlusion of the left subclavian artery and stenosis of the origin of the left vertebral artery (Figure 5). Surgical correction of the left subclavian stenosis results in disappearance of symptoms through one year of follow-up.

Discussion. At first the symptoms were believed to be of cardiac origin. The decreased blood pressure in the left arm pointed to the possibility of concomitant cerebrovascular disease, and the findings at arteriography readily explained the cause of the brain-stem ischemia. The first manifestation of cerebrovascular disease frequently occurs at the time of onset of cardiac disease. Transient or permanent decreases in cardiac output may give rise to cerebrovascular symptoms of minor to catastrophic degree. Conversely, a stroke may be the first evidence of certain types of cardiac disease, such as a "silent" myocardial infarction. Both systems should therefore be evaluated simultaneously.

CASE 5. A 63-year-old retired engineer was admitted with a history of left hemiparesis of 24 hours' duration. He had had transient episodes of left hemiparesis that had begun eight months previously. The blood pressure was 100/70 mm of mercury in both arms. Neck pulses were normal and no bruits were present. Bilateral arteriograms of the carotid artery demonstrated a filling defect at the bifurcation of the right carotid artery and a smaller filling defect in the middle cerebral artery on the same side (Figure 6). The patient's condition deteriorated steadily after admission. He was comatose when arteriograms were obtained. Embolectomy was done immediately after arteriography, but he died a month after admission.

Discussion. The patient's symptoms were caused by acute embolism at the carotid bifurcation. The origin of the embolus was presumed to be the heart. Before arteriographic examination the tentative diagnosis was atherosclerotic occlusion of the cerebral vessels. Arteriography revealed an unequivocal embolus at the carotid bifurcation. If the lesion had not been catastrophic and the patient had recovered, more attention would have been directed toward the cardiac disease and proper therapy to prevent further embolism.

CASE 6. A 59-year-old building inspector was admitted because of weakness of three day's duration in the left side of the face and the left arm and leg. Blood pressure was 160/100 mm of mercury in both arms. Neck pulsations were normal and no bruits were noted. He had mild weakness of the left side of the face, left arm and left leg. Arteriograms of the right and left subclavian and carotid arteries were obtained. No occlusive disease was seen and the only significant finding was a large intracranial aneurysm (Figure 7). Although surgery was recommended the risks were emphasized and the patient declined further treatment.

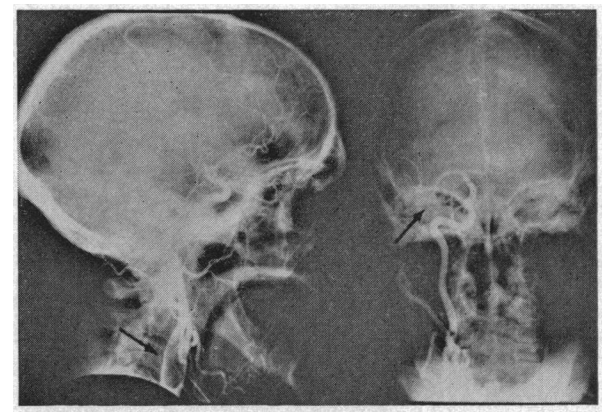

Figure 6 (Case 5).—Large embolus at the bifurcation of the right common carotid artery and small embolus in the right middle cerebral artery. (The heart was the presumed site of origin.)

Discussion. Angiography had not been performed previously in this patient because of his age and evidence of systemic arteriosclerosis. This made the diagnosis of an atherosclerotic occlusive lesion most likely. Once the pathologic findings were demonstrated, however, craniotomy was the recommended treatment of this dramatic aneurysm.

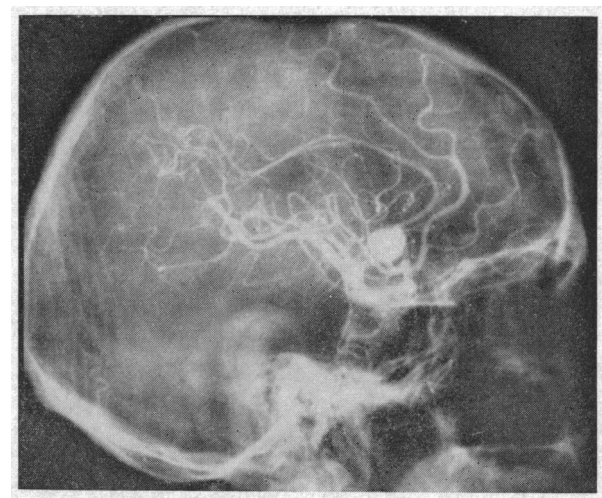

Figure 7 (Case 6).—Large aneurysm of the right anterior cerebral artery, not suspected before angiography.

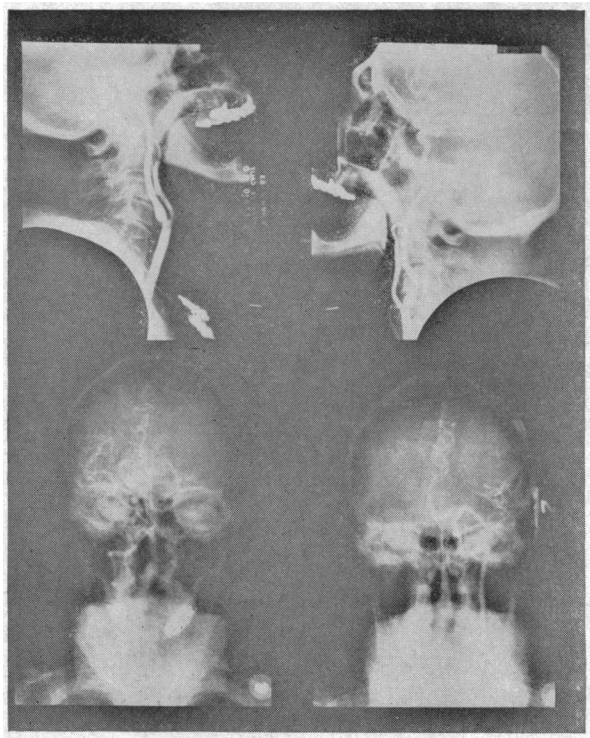

Figure 8 (Case 7).—At top are preadmission lateral views of right and left carotid arteries, showing stenotic lesions at the origin of both internal carotid arteries.
Films at bottom, repeated arteriograms including anterior-posterior views show shift of anterior cerebral arteries to the right and depression of the left middle cerebral artery consistent with a frontoparietal brain tumor.

CASE 7. A 70-year-old retired man was admitted for surgical treatment of carotid stenosis. Right hemiparesis had occurred seven days previously. Carotid arteriograms done before admission showed lesions at the origin of both of the internal carotid arteries (Figure 8). On neurologic examination the patient was alert but had flaccid hemiparesis on the right side. Carotid arteriograms were repeated and a shift in the anterior cerebral artery and depression of the middle cerebral artery, consistent with a left frontoparietal tumor, were noted (Figure 8). Craniotomy was performed a month after admission and an inoperable glioblastoma multiforme was found. The patient became progressively worse and died a month later.

Discussion. During previous examination of this patient, lesions at the origin of both internal carotid arteries were found. Because of the progression of symptoms and inadequate intracranial visualization in cerebral arteriograms taken before referral, a second arteriogram was taken which showed an unequivocal space-occupying lesion.

Atherosclerotic lesions of cerebral vessels are common and cerebrovascular occlusive disease may be found in patients whose symptoms are due to other lesions. Despite expert neurologic consultation, approximately 2 per cent of the patients in our series eventually were found to have brain tumors. In one instance, vascular reconstruction was actually done before the tumor was recognized. Satisfactory cerebral arteriograms which permit good intracranial visualization are of great value in detecting unsuspected disease. One must be constantly alert to the fact that lesions other than arteriosclerosis may be present. If the pathologic findings do not correlate with the symptoms, critical reassessment of the patient is required.

Discussion

Until recently, stroke was thought to be caused by occlusion of small arteries supplying the ischemic area of the brain.[7] In the present series, evidence of atheroma was rarely found in these smaller arteries. Careful clinical evaluation can localize the symptomatic area of the brain and the area of maximum ischemia, but locating the specific artery or arteries responsible for the patient's symptoms is much more difficult. The case reports illustrate the variety of clinical symptoms found in cerebrovascular disease as well as the variability of the findings and the frequency of multiple vessel involvement. Case 1 was a typical example of the far advanced stage that arteriosclerotic disease can reach without major localizing symptoms. In similar circumstances, an ischemic focus could occur anywhere in the brain, depending upon the relative adequacy of collateral vessels to any one area.

Even when the occlusion is within the intracranial vessels, the source of the disease is frequently the larger proximal vessels.[6] In Case 2 the significant lesion was the relatively unobtrusive plaque in the carotid artery in the neck. The stroke in this case resulted from slough of an atherosclerotic plaque with embolic occlusion of the middle cerebral artery.

Cerebral angiography may not always be successful in locating the lesion responsible for stroke. These lesions may disappear without a trace within days or weeks following the acute occlusive episode. Case 3 was an excellent example of a major stroke caused by occlusion of the internal carotid artery but which follow-up arteriography showed to have resolved completely. Smaller fibrin emboli may also cause transient ischemic attacks. Objec-

tive evidence for the presence of the lesions is the observation of fibrin emboli in the retinal arteries which disappear on serial observation.[4]

Arteriography may reveal pathologic changes other than atherosclerosis as the cause of symptoms. In Case 5, the heart was found to be the source of the embolus. However, cerebral arteriosclerosis is common, and under normal circumstances lesions may produce symptoms as a result of hemodynamic changes due to changes in posture, blood pressure or cardiac output.[2,8] In addition, one must be on his guard lest symptoms caused by brain tumor, aneurysm or other vascular malformations be attributed to atherosclerosis after incomplete visualization of the intracranial vasculature.

These cases indicate how therapy can be much more specific and effectively directed if the nature of the pathologic process is verified. When a lesion is apparently the result of hemorrhage under an atherosclerotic plaque, treatment with anticoagulants would be ineffective, nor would such agents aid in treatment of smooth stenotic lesions or of lesions that are complete and stable. Obviously, anticoagulant therapy would be indicated for embolic lesions and surgical therapy for brain tumors, aneurysms and certain extracranial occlusive lesions.

In spite of the advantages of precise diagnosis obtainable with cerebral angiography, the disadvantages—morbidity and mortality—must be considered. In light of the serious implications of stroke the minor hazards that are entailed, the discomfort and the cost of the procedure are all acceptable burdens.[11] All the arteriograms in this series were done by surgical residents in the second, third, or fourth year of training. They were responsible for all angiography performed on an active vascular service over a four-month period. This experience is probably typical of any well-equipped, well-staffed hospital which does these procedures frequently. If responsibility for the examination lay regularly and permanently with one or two experienced persons, the risk of the procedure would probably be even less. However, when the serious implications of the disease are considered, the mortality rate of 0.67 per cent and the major neurologic complication rate of 2.3 per cent are quite low. During the period of this study, a similar number of patients in whom angiography was not performed had major neurologic complications or death from stroke. In addition, the indirect benefits of angiography in the diagnosis of unsuspected lesions such as brain tumors, aneurysms and cardiac emboli alone seem to outweigh the risk. Angiography permits a clearer, more concise understanding of the cause of stroke, permits the intelligent application of therapy and can be accomplished with little risk to the patient.

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